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Database: US Patents Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: polio$ same (helicobacter or hpylori or pylori or pylor or pyloris or pyloridis) not (l1 or l2 or l3) Clear

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DB Name	Query	Hit Count	Set Name
USPT,JPAB,EPAB,DWPI	polio$ same (helicobacter or hpylori or pylori or pylor or pyloris or pyloridis) not (l1 or l2 or l3)	12	L5
USPT,JPAB,EPAB,DWPI	polio$ same (helicobacter or hpylori or pylori or pylor or pyloris or pyloridis) not l1 or l2 or l3	28	L4
USPT,JPAB,EPAB,DWPI	l2 not l1	11	L3
USPT,JPAB,EPAB,DWPI	polio$.ti,ab,clm. and helicobacter	16	L2
USPT	polio$.ti,ab,clm. and helicobacter	5	L1

Helicobacter **-specific cell-mediated immune responses display a predominant** Th1 **phenotype and promote a delayed-type hypersensitivity response in the stomachs of mice.**

Mohammadi M; Czinn S; Redline R; Nedrud J
Institute of Pathology, Case Western Reserve University, Cleveland, OH 44106, USA.

Journal of immunology (UNITED STATES) Jun 15 1996, 156 (12) p4729-38, ISSN 0022-1767 Journal Code: IFB
Contract/Grant No.: DK 46461, DK, NIDDK; HL 37117, HL, NHLBI
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9609
Subfile: AIM; INDEX MEDICUS

Studies regarding the nature of cell-mediated immunity in **Helicobacter** pylori infection and its role in pathogenesis have yielded controversial results. To address this issue in a controlled manner, we have employed the well-characterized **Helicobacter** felis-mouse model. Immunized/challenged and nonimmunized/infected mice were evaluated for cellular proliferation, gastric inflammation, and cytokine and Ab production at various times after infection. We observed two types of cell-mediated immune responses depending on the nature of the Ag preparation. The first response is a **Helicobacter** -independent response, present in all experimental groups, which is directed toward Ags such as urease and heat shock proteins. The second is a **Helicobacter** -dependent cellular response restricted to mice previously exposed to **Helicobacter** Ags either by immunization or infection. This response was not seen in noninfected controls. The **Helicobacter** -dependent cellular response had a Th1 phenotype, as either infected or immunized/challenged mice demonstrated local and systemic production of IFN-gamma and undetectable levels of IL-4 or IL-5. Cellular proliferation correlated with the severity of gastric inflammation in both immunized/challenged (protected) and nonimmunized/infected mice. Finally, in vivo neutralization of IFN-gamma resulted in a significant reduction of gastric inflammation in H. felis-infected, as well as immunized/challenged, mice. This treatment also revealed the presence of Th2 cells, restricted to immunized/challenged mice, as demonstrated by local and systemic production of IL-4 in these mice. These data demonstrate that **Helicobacter** infection and/or immunization stimulate a predominantly Th1-type, Ag-specific response and promote a local delayed-type hypersensitivity response in the stomach that may be inhibited by depletion of IFN-gamma.

Tags: Animal; Female; Support, U.S. Gov't, P.H.S.
Descriptors: **Helicobacter** --Immunology--IM; *Hypersensiti

Murine CD4 T-cell response to Helicobacter **infection: TH1 cells enhance gastritis and TH2 cells reduce bacterial load.**

Mohammadi M; Nedrud J; Redline R; Lycke N; Czinn SJ

Institute of Pathology, Case Western Reserve University, Cleveland, Ohio, USA.

Gastroenterology (UNITED STATES) Dec 1997, 113 (6) p1848-57, ISSN 0016-5085 Journal Code: FH3

Contract/Grant No.: DK-46461, DK, NIDDK; AI 40701-01, AI, NIAID; AI-36359, AI, NIAID

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9803

Subfile: AIM; INDEX MEDICUS

BACKGROUND & AIMS: Previous findings suggest that TH1 cellular immune responses contribute to **Helicobacter** -associated gastritis. To further investigate this issue, interleukin 4 gene targeted mice were infected with **Helicobacter** felis, and a series of adoptive transfer experiments was performed to evaluate the role of both TH1 and TH2 cells. METHODS: Antigen-specific spleen cells from immunized/challenged or nonimmunized/infected mice or CD4+ T-cell lines were transferred adoptively into naive recipients before live bacterial challenge. RESULTS: Transfer of cells from both groups of donors as well as TH1 or TH2 cell lines exacerbated gastric inflammation in the recipients. No effect on bacterial load was observed in recipients of bulk spleen cells from infected mice or recipients of TH1 cell lines. In contrast, when either a TH2 cell line or bulk cells from immunized challenged mice were transferred adoptively, recipients showed a dramatic reduction in bacterial load. Increased numbers of bacteria were also noted in interleukin 4-deficient mice. CONCLUSIONS: These data suggest a differential contribution of TH1 and TH2 cell-mediated immune responses in **Helicobacter** infection: one associated with the pathogenesis of disease (TH1 phenotype) and the other associated with protection from or control of infection (TH2 phenotype).

Tags: Animal; Female; Support, U.S. Gov't, P.H.S.

Th1/**Th2 cells.**

Romagnani S
Department of Internal Medicine, University of Florence, Italy.

Inflammatory bowel diseases (UNITED STATES) Nov 1999, 5 (4) p285-94,
ISSN 1078-0998 Journal Code: C2I
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 0002
Subfile: INDEX MEDICUS

A large body of evidence indicates the existence of functionally polarized CD4+ T-cell responses based on their profile of cytokine secretion. Type 1 T helper (Th1) cells produce interferon-gamma, interleukin (IL)-2, and tumour necrosis factor (TNF)-beta, which activate macrophages and are responsible for cell-mediated immunity and phagocyte-dependent protective responses. By contrast, type 2 Th (Th2) cells produce IL-4, IL-5, IL-10, and IL-13, which are responsible for strong antibody production, eosinophil activation, and inhibition of several macrophage functions, thus providing phagocyte-independent protective responses. Th1 cells mainly develop following infections by intracellular bacteria and some viruses, whereas Th2 cells predominate in response to infestations by gastrointestinal nematodes. Polarized Th1 and Th2 cells not only exhibit different functional properties, but also show the preferential expression of some activation markers and distinct transcription factors. Several mechanisms may influence the Th cell differentiation, which include the cytokine profile of "natural immunity" evoked by different offending agents, the nature of the peptide ligand, as well as the activity of some costimulatory molecules and microenvironmentally secreted hormones, in the context of the individual genetic background. In addition to playing different roles in protection, polarized Th1-type and Th2-type responses are also responsible for different types of immunopathological reactions. Th1 cells are involved in the pathogenesis of organ-specific autoimmune disorders, Crohn's disease, **Helicobacter** pylori-induced peptic ulcer, acute kidney allograft rejection, and unexplained recurrent abortions. In contrast, allergen-specific Th2 responses are responsible for atopic disorders in genetically susceptible individuals. Moreover, Th2 responses against still unknown antigens predominate in Omenn's syndrome, idiopathic pulmonary fibrosis, and progressive systemic sclerosis. Finally, the prevalence of Th2 responses may play some role in a more rapid evolution of human immunodeficiency virus infection to the full-blown disease. The Th1/Th2 paradigm also provides the rationale for the development of new types of vaccines against infectious agents and of novel strategies for the therapy of allergic and autoimmune disorders. (131 Refs.)

Tags: Animal; Human

Antrum- and corpus mucosa-infiltrating CD4(+) lymphocytes in Helicobacter **pylori gastritis display a** Th1 **phenotype.**

Sommer F; Faller G; Konturek P; Kirchner T; Hahn EG; Zeus J; Rollinghoff M; Lohoff M

Institut fur Klinische Mikrobiologie, Immunologie und Hygiene der Universitat Erlangen-Nurnberg, Erlangen, Germany. sommer@mikrobio.med.uni.erlangen.de

Infection and immunity (UNITED STATES) Nov 1998, 66 (11) p5543-6, ISSN 0019-9567 Journal Code: GO7

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9901

Subfile: INDEX MEDICUS

In this study, cytokine patterns produced by CD4(+) T cells isolated from antrum or corpus gastral biopsy specimens of 10 patients with **Helicobacter** pylori-positive gastritis were compared. To this end, expression of intracellular cytokines (interleukin-4 [IL-4] and gamma interferon) and of CD4 was assessed by flow cytometry. Ten to 60% of the isolated CD4(+) T cells produced gamma interferon upon stimulation. With the exception of one patient, IL-4-positive CD4(+) cells were not detected. Therefore, CD4(+) cells infiltrating antrum and corpus stomach mucosa during H. pylori infection show a Th1 phenotype. This polarized Th1-type response may contribute to the inability of the immune system to eradicate H. pylori infection.

Tags: Female; Human; Male; Support, Non-U.S. Gov't

Descriptors: CD4-Positive T-Lymphocytes--Immunology--IM; *Gastric Mucosa --Immunology--IM; *Gastritis--Immunology--IM; * **Helicobacter** pylori --Immunology--IM; ***Helicobacter** Infections--Immunology--IM; Adult; Aged; Aged, 80 and over; Gastric Mucosa--Pathology--PA; Gastritis--Pathology--PA; **Helicobacter** pylori--Pathogenicity--PY; Middle Age; Phenotype; Pyloric Antrum--Immunology--IM; Pyloric Antrum--Pathology--PA; Th1 Cells --Chemistry--CH; Th1 Cells--Immunology--IM

Chronic active hepatitis induced by Helicobacter **hepaticus in the A/JCr mouse is associated with a** Th1 **cell-mediated immune response.**

Whary MT; Morgan TJ; Dangler CA; Gaudes KJ; Taylor NS; Fox JG

Division of Comparative Medicine, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139, USA. mwhary@mit.edu

Infection and immunity (UNITED STATES) Jul 1998, 66 (7) p3142-8, ISSN 0019-9567 Journal Code: GO7

Contract/Grant No.: RO1 CA 67529, CA, NCI; RO1 DK 52413, DK, NIDDK; RR 07036, RR, NCRR

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9809

Subfile: INDEX MEDICUS

Helicobacter hepaticus infection in A/JCr mice results in chronic active hepatitis characterized by perivascular, periportal, and parenchymal infiltrates of mononuclear and polymorphonuclear cells. This study examined the development of hepatitis and the immune response of A/JCr mice to H. hepaticus infection. The humoral and cell-mediated T helper immune response was profiled by measuring the postinfection (p.i.) antibody response in serum, feces, and bile and by the production of cytokines and proliferative responses by splenic mononuclear cells to H. hepaticus antigens. Secretory immunoglobulin A (IgA) and systemic IgG2a antibody developed by 4 weeks p.i. and persisted through 12 months. Splenocytes from infected mice proliferated and produced more gamma interferon (IFN-gamma) than interleukin-4 (IL-4) or IL-5 when cultured with H. hepaticus outer membrane proteins. The predominantly IgG2a antibody response in serum and the in vitro production of IFN-gamma in excess of IL-4 or IL-5 are consistent with a Th1 immune response reported in humans and mice infected with **Helicobacter** pylori and **Helicobacter** felis, respectively. Mice infected with H. hepaticus developed progressively severe perivascular, periportal, and hepatic parenchymal lesions consisting of lymphohistiocytic and plasmacytic cellular infiltrates. In addition, transmural typhlitis was observed at 12 months p.i. The characterization of a cell-mediated Th1 immune response to H. hepaticus infection in the A/JCr mouse should prove valuable as a model for experimental regimens which manipulate the host response to **Helicobacter** .

Tags: Animal; Male; Support, U.S. Gov't, P.H.S.

Analysis of TH1 **and TH2 cytokine production in low grade B cell gastric MALT-type lymphomas stimulated in vitro with** Helicobacter **pylori.**

Hauer AC; Finn TM; MacDonald TT; Spencer J; Isaacson PG

Department of Paediatric Gastroenterology, St Bartholomew's School of Medicine, St Bartholomew's Hospital, London, UK.

Journal of clinical pathology (ENGLAND) Nov 1997, 50 (11) p957-9, ISSN 0021-9746 Journal Code: HT3

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9804

Subfile: AIM; INDEX MEDICUS

Previous studies have suggested that the dependence of low grade B cell gastric lymphoma on infection of the gastric mucosa with **Helicobacter** pylori results from help provided by H pylori specific tumour infiltrating T cells. ELISPOT analysis was used to characterise functional subpopulations of tumour infiltrating T cells. The production of the TH1 cytokine interferon gamma and TH2 cytokines interleukin (IL)-4, IL-5, and IL-10 were measured in tumour cell suspensions from two cases of low grade B cell gastric lymphoma, one case of thyroid gland lymphoma, and one case of salivary gland lymphoma. Cells were assayed on day 0 and following 24 hours incubation either in culture medium or with a range of strains of H pylori. There was a dominant TH1-type (pro-inflammatory) response consistent with the TH1 response observed in H pylori gastritis.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: Antigens, Bacterial--Immunology--IM; *Cytokines --Biosynthesis--BI; * **Helicobacter** pylori--Immunology--IM; *Lymphoma,

Helicobacter **-specific cell-mediated immune responses display a predominant** Th1 **phenotype and promote a delayed-type hypersensitivity response in the stomachs of mice.**

Mohammadi M; Czinn S; Redline R; Nedrud J
Institute of Pathology, Case Western Reserve University, Cleveland, OH 44106, USA.

Journal of immunology (UNITED STATES) Jun 15 1996, 156 (12) p4729-38, ISSN 0022-1767 Journal Code: IFB
Contract/Grant No.: DK 46461, DK, NIDDK; HL 37117, HL, NHLBI
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9609
Subfile: AIM; INDEX MEDICUS

Studies regarding the nature of cell-mediated immunity in Helicobacter pylori infection and its role in pathogenesis have yielded controversial results. To address this issue in a controlled manner, we have employed the well-characterized Helicobacter felis-mouse model. Immunized/challenged and nonimmunized/infected mice were evaluated for cellular proliferation, gastric inflammation, and cytokine and Ab production at various times after infection. We observed two types of cell-mediated immune responses depending on the nature of the Ag preparation. The first response is a Helicobacter-independent response, present in all experimental groups, which is directed toward Ags such as urease and heat shock proteins. The second is a Helicobacter-dependent cellular response restricted to mice previously exposed to Helicobacter Ags either by immunization or infection. This response was not seen in noninfected controls. The Helicobacter-dependent cellular response had a Th1 phenotype, as either infected or immunized/challenged mice demonstrated local and systemic production of IFN-gamma and undetectable levels of IL-4 or IL-5. Cellular proliferation correlated with the severity of gastric inflammation in both immunized/challenged (protected) and nonimmunized/infected mice. Finally, in vivo neutralization of IFN-gamma resulted in a significant reduction of gastric inflammation in H. felis-infected, as well as immunized/challenged, mice. This treatment also revealed the presence of Th2 cells, restricted to immunized/challenged mice, as demonstrated by local and systemic production of IL-4 in these mice. These data demonstrate that Helicobacter infection and/or immunization stimulate a predominantly Th1-type, Ag-specific response and promote a local delayed-type hypersensitivity response in the stomach that may be inhibited by depletion of IFN-gamma.

Tags: Animal; Female; Support, U.S. Gov't, P.H.S.
Descriptors: *Helicobacter--Immunology--IM; *Hypersensitivity, Delayed --Immunology--IM; *Lymphocyte Transformation; *Stomach--Immunology--IM; *Th1 Cells--Immunology--IM; Gastritis--Immunology--IM; Immunity, Cellular; Interferon Type II--Physiology--PH; Mice; Mice, Inbred C57BL; Urease --Immunology--IM
CAS Registry No.: 82115-62-6 (Interferon Type II)
Enzyme No.: EC 3.5.1.5 (Urease)

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L5: Entry 12 of 12 File: DWPI Sep 18, 1997

DERWENT-ACC-NO: 1997-470645
DERWENT-WEEK: 199743

TITLE: Enhancing an immune response by using dialdehyde compounds - as crosslinkers and immunostimulatory agents, useful against antigens, cancers, tumours or infections caused by pathogens

PRIORITY-DATA: 1997US-0803764 (February 21, 1997), 1996US-0616834 (March 15, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 9733612 A1	September 18, 1997	E	106	A61K039/00
AU 9719910 A	October 1, 1997	N/A	000	A61K039/00

INT-CL (IPC): A61K 39/00; A61K 39/02; A61K 39/12; A61K 39/39

TITLE-TERMS: ENHANCE IMMUNE RESPOND COMPOUND CROSSLINK AGENT USEFUL ANTIGEN CANCER TUMOUR INFECT CAUSE PATHOGEN

DERWENT-CLASS: B02 B03 B04 D16

CPI-CODES: B04-B03B; B14-A01; B14-A02; B14-A03; B14-A04; B14-G01; D05-H07; D05-H10;

CHEMICAL-CODES:< pre> Chemical Indexing M1 *01* Fragmentation Code M423 M781 M903 P001 P210 P434 P633 Q233 V791

SECONDARY-ACC-NO:
CPI Secondary Accession Numbers: C1997-149551

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L5: Entry 4 of 12 File: USPT May 16, 2000

US-PAT-NO: 6063384
DOCUMENT-IDENTIFIER: US 6063384 A

TITLE: Encapsidated recombinant viral nucleic acid and methods of making and using same

DATE-ISSUED: May 16, 2000

INT-CL: [7] C12P 21/02, C12N 15/43, A61K 39/13
US-CL-ISSUED: 424/199.1; 424/217.1, 424/208.1, 435/69.3, 435/172.1, 435/320.1
US-CL-CURRENT: 424/199.1; 424/208.1, 424/217.1, 435/320.1, 435/69.3
FIELD-OF-SEARCH: 424/199.1, 424/217.1, 424/208.1, 435/69.3, 435/172.1, 435/320.1